that this is established recommended practice. An assurance was added that no harm would follow, provided environmental conditions did not encourage the dissemination of spores.

Remote from the bedside the euphemism for saturation resulting from incontinence is "condensation," and it has, apparently, long been known that the covers of certain best buy NHS mattresses provide uncertain protection against this and other forms of pollution. The mattresses themselves cannot be cleaned and are too costly to be destroyed. I am advised that it would be unhelpful to tell unsuspecting patients that when the beds they occupy become malodorous it is due to "natural condensation." It might offend the critical value—productivity divided by expenditure—in a business struggling desperately for survival and having already abandoned any pretensions of propriety.

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Hypophosphataemia in acute liver failure

SIR,—We can confirm and extend the finding of hypophosphataemia after paracetamol induced fulminant hepatic failure reported by Dr D J Dawson and colleagues (21 November, p 1312).

We reviewed data from 15 patients with acute hepatorenal failure after paracetamol self poisoning. There were nine women and six men with a mean age of 31 years (range 20-46); their mean prothrombin ratio was 4·9 (range 1·4-12·6) and serum creatinine concentration 468 μmol/l (range 67-817) on admission. Twelve were in grade 4 hepatic encephalopathy; 14 had a metabolic acidaemia on admission (base excess ≥18 mmol/l); 10 were hypokalaemic (serum potassium <3·5 mmol/l); and five were hypophosphataemic (serum phosphate <0·8 mmol/l); a further three subsequently developed hypophosphataemia. Seven patients died.

Several differences from the previous report emerge from our data. A serum creatinine concentration of >250 μmol/l did not prevent hypophosphataemia in six out of 12 patients. The cause of this hypophosphataemia is doubtless multifactorial; on admission it is probably due to metabolic acidosis secondary to tissue hypoxia.[1] Intracellular organophosphorus compounds break down in the presence of intracellular acidosis, and phosphate is lost in the urine.[2] After admission hypophosphataemia may develop because of the correction of the acid base state, the use of dextrose infusions to maintain euglycaemia,[3] and renal tubular damage.[4] Phosphate losses, however, may also occur because of the treatments undertaken for fulminant hepatic failure, of which high flux haemofiltration is perhaps the most important. Haemofiltration has been used recently to manage patients with fulminant hepatic failure, and ultrafiltrate losses are usually replaced with commercially available electrolyte replacement solutions which are phosphate free. Four of the authors' patients received haemofiltration, and in three the serum phosphate value fell further while the fourth remained severely hypophosphataemic. Only 15% of their patients required phosphate supplements, suggesting that in most patients the changes reflected intracellular shifts rather than total body depletion. Among our patients, however, most of those who developed hypophosphataemia (75%) also showed hypokalaemia,[5] suggesting, in the presence of a metabolic acidaemia (anion gap mean 22±2), that the intracellular pools were also reduced. Haemofiltration has become our preferred treatment in patients with hepatorenal failure.[6] We add potassium phosphate to potassium free haemofiltration replacement solution, both to correct hypophosphataemia on admission and to prevent its occurrence during treatment.

The role of hypophosphataemia in the development of hepatic coma is conjectural, but one of our cases suggests that it may be important.

A 40 year old woman was transferred in grade 3 coma with fulminant hepatic failure of unknown cause on a dextrose infusion regimen. On arrival she was conscious, but after two hours she suddenly deteriorated into grade 4 coma. She was ventilated and an intracranial pressure monitor was inserted. She subsequently suffered a cardiac arrest due to hypokalaemia (serum potassium 1·3 mmol/l, having been 2·2 mmol/l before transfer). She was resuscitated, supported with potassium and dextrose infusions, and started on continuous haemofiltration at an ultrafiltration rate of 1000 ml/h, the losses being replaced with commercially available replacement solution. She remained hypotensive with a normal intracranial pressure but suffered an asystolic cardiac arrest seven hours later when the serum potassium had been stabilised at 2·9 mmol/l. Serum phosphate estimations became available only after death; before transfer the value was 1·08 mmol/l, before the first cardiac arrest 0·23 mmol/l, and immediately before asystole 0·05 mmol/l. Both serum calcium and magnesium concentrations were normal. This patient was phosphate deficient. We believe that the neurological deterioration was related to the fall in the serum phosphate value and that death ensued because of the reduced phosphate value.

The practical implications of these findings are that acute renal failure and hypophosphataemia may coexist (often with hypokalaemia), that phosphate replacement should be considered during haemofiltration, and, perhaps most important, that clinicians should expect to request, and biochemists to provide, acute serum phosphate measurements in centres dealing with these metabolically complex patients.

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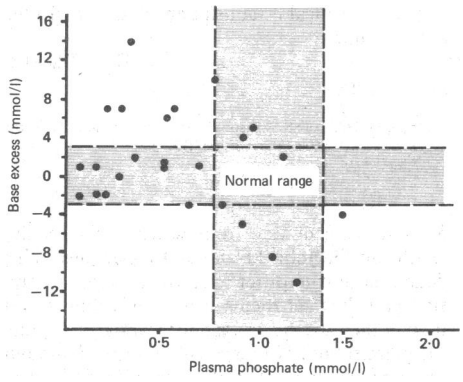
SIR,—Dr D J Dawson and others (21 November, p 1312) highlight the occurrence and frequency of hypophosphataemia in acute liver failure and the expected negative correlation with renal failure. The recognition of hypophosphataemia as a complication of acute liver failure and the concept that hypophosphataemia may be implicated in hepatic encephalopathy, however, are not new, having been described as early as 1972 by Knell and others.[1] They investigated 26 patients with acute liver failure and showed hypophosphataemia in 16. A negative correlation with base excess suggested that respiratory alkalosis was a contributory factor in its pathogenesis (figure). This frequency (62%) is identical with that reported by Dr Dawson and his colleagues.

In a recent further study we again found the same frequency (60%) of hypophosphataemia in 30 consecutive patients admitted to the liver failure unit in grade 3 or 4 coma. Survival in the seven patients with severe hypophosphataemia (<0·4 mmol/l) was similar to that in the other 23 patients (4 (57%) and 11 (47%) respectively). Although survival was similar in those patients with and those without hypophosphataemia, we would endorse the recommendation that serum phosphate concentrations should be measured routinely in this condition and severe hypophosphataemia corrected with intravenous phosphate.



Relation between hypophosphataemia and base excess. From Knell *et al*.[1]

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Responding flexibly but not gullibly to drug addiction

SIR,—The leading article by Dr John Strang and colleagues (28 November, p 1364) emphasising the need to respond to the individual addict rather than to the stereotype opens an unexpected door to reality. Furthermore, its encouragement of general practitioners to provide treatment to addicts is what the Association of Independent Doctors in Addiction has been saying all along.

It quotes a follow up study of those addicts who had been turned away by the clinics, which showed that all of them went on supporting and nourishing the criminal black market, though about half of them to a lesser extent than before.[1] The damage done to society by this black market in heroin is difficult to exaggerate. The Mafia's way into Britain to control this black market was cleared years ago with a change of policy by the clinics from one of relatively liberal prescribing to a rigid refusal to prescribe injectable drugs.

It is ironic that this leading article of hope should appear just at the time that Dr Ann Dally's persecution for flexible prescribing (having by this means weaned off drugs some dozens of injecting addicts of 10 to 30 years' standing) reaches new lengths. After the General Medical Council forbade her to prescribe any more controlled drugs to her addict patients she tried to get them taken on by the various NHS treatment centres and prescribed minor tranquillisers or painkillers such as a benzodiazepine or dihydrocodeine until they could be seen. For those patients who were turned down by the clinics and also for those who could not bring themselves to reattend particular clinics she did the same.

Now, I understand, the police have been asked to bring a criminal prosecution against her for such prescribing. (How many doctors know that a benzodiazepine is technically a controlled drug?) It is incomprehensible to me except in the context of